Pharmacological Actions of Oxyethylammonium Methylphenoxyacetate (Trekrezan)

Oxyethylammonium methylphenoxyacetate, commonly known as Trekrezan, is an adaptogenic immunomodulator with a broad spectrum of effects. It acts primarily by stimulating interferon production, enhancing immune responses, stabilizing cellular energy metabolism, and providing anti-toxic, anti-inflammatory, and anti-hypoxic properties. Below, I outline its key pharmacological actions, including available quantitative values where possible (drawn from clinical studies, animal models, and mechanistic data). These values indicate the influence of each action, such as magnitude of effect, time course (e.g., onset or duration), and impact on biological markers or outcomes. Note that much of the quantitative data comes from rat models or specific clinical contexts (e.g., ARVI or post-COVID asthenia), as human pharmacokinetic and pharmacodynamic studies are limited.

1. **Immunomodulatory and Antiviral Action**

- **Mechanism**: Stimulates production of α- and γ-interferons, activates cellular and humoral immunity, and enhances phagocytic activity of macrophages. This leads to improved immune status and antiviral defense.
- **Quantitative Values and Influence**:
    - Reduces ARVI duration by an average of 2.2 days (observational study in 130 adults; faster symptom resolution in treatment group vs. control, with significant reduction in fever, weakness, and fatigue by days 3–5).
    - In post-COVID-19 asthenia (study in patients recovering from mild/moderate COVID-19): Eliminates asthenia symptoms in 80% of patients (severe asthenia reduced from 20% to 0%; moderate asthenia from 70–73% to 0% after 8-day course; improves emotional well-being and sleep quality vs. control).
    - In rat parodont inflammation model (intraperitoneal administration): Elevates lymphokine-producing function of lymphocytes by 67% (with concanavalin A) and 73% (with phytohemagglutinin); increases phagocyte activity of neutrophils by 24%; decreases phagocyte number and phagocytosis index by 26% and 14%, respectively (indicating normalized immune response within 3 days).
    - In rat bronchopneumonia model (25 mg/kg dose): Normalizes lymphocyte immune status (e.g., restores spot-forming activity of stem cells) and stimulates lympho- and hemopoiesis within days of administration.
    - In vitro: Stimulates mononuclear cell proliferation via direct action on B lymphocytes and increased lymphokine/monokine production (steady effect observed).
- **Time Course**: Rapid onset (interferon induction within 6–24 hours after single dose); long-term antiviral effect (up to 8 days in ARVI treatment). Influences by accelerating recovery from viral infections and preventing complications.

2. **Adaptogenic Action**

- **Mechanism**: Increases resistance to stress, hypoxia, temperature extremes, and environmental factors; enhances physical and mental endurance.
- **Quantitative Values and Influence**:
    - In clinical use: Improves working capacity in asthenia (including post-COVID); reduces symptoms of fatigue and stress (e.g., in high intellectual/physical load periods, with effects lasting 7–14 days on a standard course).
    - In rat hypoxic hypoxia model (25 mg/kg over 3 days): Increases adaptive metabolic changes in brain, heart, and liver (e.g., enhances tissue resistance to oxygen deprivation).
    - In extreme conditions (e.g., climate changes, overload): Demonstrated effectiveness in clinical trials, with no specific % values but noted as "significantly higher efficiency" than natural adaptogens like ginseng.
- **Time Course**: Effects build over 7–14 days (prevention courses); influences by boosting overall resilience, reducing recovery time from stressors.

3. Anti-Inflammatory Action

- **Mechanism**: Reduces oxidative stress and inflammation markers; normalizes immune responses in inflammatory models.
- **Quantitative Values and Influence**:
    - In rat parodont inflammation model: Decreases malonic dialdehyde (lipid peroxidation marker) by 61%; dienic conjugates by 68%; increases recovered glutathione by 2.9-fold and superoxide dismutase activity by 79% (within 3 days, leading to anti-inflammatory resolution).
    - In rat bronchopulmonary inflammation model (25 mg/kg): Produces steady anti-inflammatory activity; less pronounced than energy-stabilizing effects but normalizes immune status.
    - In eye injury patent (5% solution): Accelerates corneal healing (subconjunctival injections/applications; no specific % but noted as effective for mechanical injuries).
- **Time Course**: Observable within 3 days in models; influences by mitigating tissue damage and promoting regeneration.

4. Energy-Stabilizing (Metabolic) Action

- **Mechanism**: Normalizes cellular energy metabolism by reducing anaerobic byproducts and enhancing ATP production.
- **Quantitative Values and Influence**:
    - In rat bronchopneumonia model (25 mg/kg): Decreases lactate, ADP, and AMP levels; increases pyruvate and ATP in blood lymphocytes and lung tissue (normalization within treatment period, stabilizing energy for immune function).
    - In rat inflammation model: Similar reductions in lactate/ATP hydrolysis products and increases in pyruvate/ATP (more pronounced in lung tissue).
- **Time Course**: Effects manifest during acute treatment (e.g., 3–8 days); influences by supporting cellular function under stress, reducing fatigue.

5. Anti-Toxic Action

- **Mechanism**: Reduces toxin effects (e.g., heavy metals, ethanol, solvents); stimulates phagocyte toxin lysis and liver enzyme activity.
- **Quantitative Values and Influence**:
    - In poisoning (human): 400–600 mg/day for 5 days reduces manifestations (e.g., alcohol withdrawal syndrome).
    - In clinical review: Pronounced anti-toxic effect in ethanol/metal salt poisoning (no specific % but noted as effective detoxification).
- **Time Course**: 5-day course for acute intoxication; influences by accelerating toxin removal and symptom relief.

6. Other Actions

- Antisclerotic (rat models): Potential mechanisms explored, but no quantitative values.
- Hepatoprotective: Stimulates hepatocyte regeneration (e.g., post-hepatectomy).
- No teratogenic or adverse effects in trials; good tolerability.

Half-Life

- Mean terminal half-life: 13.39 hours (single dose); decreases to 5.11 hours with repeated dosing (possibly due to autoinduction or metabolic changes).
- No significant changes in elderly patients, children 12–18 years, or those with renal impairment.

Bioavailabilities

- **Oral Route**: Primary administration method (tablets), indicating good oral bioavailability. Apparent oral clearance: 12.27 L/h (single dose), increasing to 41.38 L/h with repeated dosing (geometric mean; suggests high absorption but potential for faster elimination over time). No absolute bioavailability percentage available, but high based on effective oral dosing and rapid metabolism in liver (excreted mainly as metabolites).
- **Other Routes**: Subconjunctival (eye drops, 5% solution for corneal healing); no specific bioavailability data. Not significantly altered in special populations.

Dosages

- **Form**: 200 mg tablets (oral).
- **ARVI Treatment (adults/children ≥12 years)**: Day 1: 600 mg (3 × 200 mg); Days 2–8: 200 mg daily (total course: 2,000 mg; 1 package of 10 tablets suffices).
- **ARVI Prevention**: 200 mg daily for 14 days.
- **Stress/Endurance/Adaptation**: Day 1: 600 mg; then 200 mg daily for 7 days.
- **Poisoning/Intoxication (e.g., heavy metals, ethanol withdrawal)**: 400–600 mg daily for ≥5 days (repeatable after 1–2 months if needed).
- **Bodybuilding/Performance**: 400 mg daily (e.g., 200 mg pre- and post-training; 7-day course).
- **Minimum Effective Dose**: 200 mg daily (prevention or maintenance; effective for immunity boost).
- **Safe Range**: Low toxicity; LD50 (rats): >3,700 mg/kg IP, >6,300 mg/kg oral; >2,000 mg/kg IP, >3,200 mg/kg oral (mice). No adverse effects in clinical trials; well-tolerated up to 600 mg/day. Avoid in children <12 years, pregnancy/lactation (insufficient data).